MRI MEASUREMENTS OF CRANIOSPINAL AND INTRACRANIAL VOLUME CHANGE IN HEALTHY AND HEAD TRAUMA CASES

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Abstract: **The volumes of the intracranial space and the craniospinal system as a whole change during the cardiac cycle. These volume changes are caused by the pulsatile arterial inflow to the cranium, venous outflow from the cranium, and cerebrospinal fluid (CSF) flow that oscillates back and forth between the cranium and the spinal canal. The volume changes can be measured accurately and reproducibly using a dynamic, motion-sensitive MRI technique [1]. It appears intuitive that the volume change of the entire craniospinal system (CSVC) should be greater than the intracranial volume change (ICVC). However, since they exhibit varying temporal information, CSVC can be smaller than ICVC. In the present study, these volume changes were measured in healthy humans and trauma cases. In the trauma cases, it was found that CSVC was smaller than ICVC. The cause was found to be increased pulsatility in the venous flow channels. It is suspected that the resulting relationship between ICVC and CSVC is related to the incidence of trauma, and perhaps CSVC being smaller than ICVC could serve as an indicator.**

Keywords: **head trauma, intracranial volume change, craniospinal volume change, craniospinal system, arterial flow, venous flow, CSF flow, modulation transfer function**

I. INTRODUCTION

The craniospinal system consists of two subcompartments, the intracranial space (skull) and the spinal canal. Cerebrospinal fluid (CSF) oscillates back and forth between these two subcompartments. The system can be modeled with arterial inflow and venous outflow. During systole, arterial blood rushes into the cranium, forcing the CSF out into the spinal canal. As the blood drains through venous channels during diastole, the CSF refills the cranium.
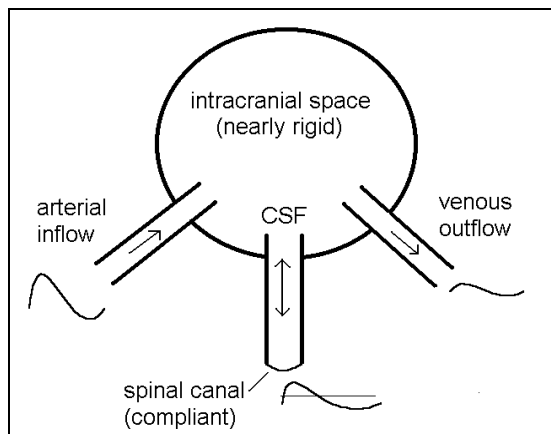

Fig. 1: The craniospinal system

Since this is a closed system, the entire craniospinal system volume change (CSVC) can be found by subtracting the venous (V) outflow from the arterial (A) inflow and examining the peak to peak change in the integral of A-V over one cardiac cycle. If the CSF flow is also subtracted from the A-V flow, the intracranial volume change (ICVC) can be obtained similarly. This method is used to measure the intracranial compliance and pressure from the ratio of volume and pressure changes that occur during the cardiac cycle [2].

II. METHOD

Arterial, venous, and CSF flows were measured using a dynamic, motion-sensitive MRI technique [1]. This technique results in cross-sectional images of velocity. This means that the pixel values are proportional to the velocity of the particles. Gray areas are static; black represents velocity in one direction, while white represents velocity in the other. In the sample image (Fig. 2), the large white spot is a jugular vein carrying blood away from the cranium while the large black spots are carotid and vertebral arteries carrying blood into the cranium. The flow rates can be obtained from the images of velocity by integrating velocity values over the lumen area. This sample image is actually 1 of 32 images that are taken per cardiac cycle. Flow rates derived from each image are plotted together to create the flow waveforms in Fig. 3 that illustrate changing flow during one heart beat.
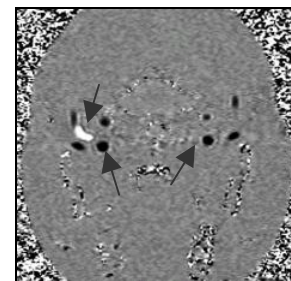

Fig. 2: A sample phase contrast MRI image

Regular biases in the MRI velocity measurements that result from baseline phase drifts are corrected by using the following constraint, which represents the conservation of fluid within the system and is known as the Monro-Kellie doctrine. This means that there is no change in the average intracranial volume over time:

$$\sum_{\substack{cardiac \\ cycle}} [A(t) - V(t) - CSF(t)]\Delta t = 0$$

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Abstract

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This method has been applied to heatlhy humans and humans who had experienced head trauma. Both the total craniospinal volume change and the intracranial volume change were measured.

III. RESULTS

The craniospinal and intracranial volume changes for 5 healthy normal subjects are shown in Table I. All measured CSVC values are greater than the measured ICVC values.

TABLE I
Healthy mean values

	CSVC	ICVC
Subject 1	1.12	0.49
Subject 2	1.14	0.30
Subject 3	0.64	0.49
Subject 4	0.73	0.38
Subject 5	1.30	0.61

Student's t-test resulted in a statistically significant difference, p<0.01.

In two cases, subject F and subject D, it was retrospectively found that they had experienced head trauma 15 and 2 years ago, respectively. For these subjects, repeated measurements of CSVC and ICVC values are shown in Tables II and III. CSVC is consistently smaller than ICVC.

TABLE II
Subject F

	CSVC	ICVC
Trial 1	0.39	0.82
Trial 2	0.30	0.50
Trial 3	0.29	0.48
Trial 4	0.36	0.61
Mean	0.34	0.60
St dev	0.05	0.16
% St dev	14	26

Student's t-test resulted in a statistically significant difference, p<0.01.

TABLE III
Subject D

	CSVC	ICVC
Trial 1	0.35	0.71
Trial 2	0.41	0.42
Mean	0.38	0.57

The measured arterial (A), venous (V), A-V, and CSF flows, as well as volume curves that are obtained from the integrals of these flows for a normal case and subject D, a trauma case, are shown in Fig. 3-5.

IV. DISCUSSION

The arterial and CSF flows are quite similar in both cases as shown in Fig. 3; however, the venous flow is markedly more pulsatile in the trauma case (Fig. 3b). This causes the A-V waveform to be negative during systole (Fig. 4b) unlike the normal case (Fig. 4a). Comparing Figs. 4a and 4b, it appears that the CSF follows the A-V to a much greater degree in a healthy subject than in a trauma subject.
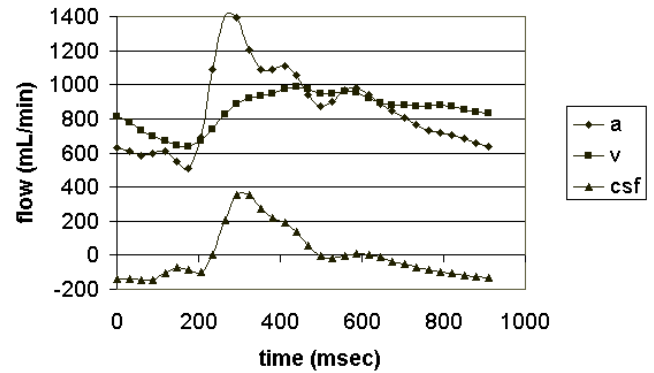
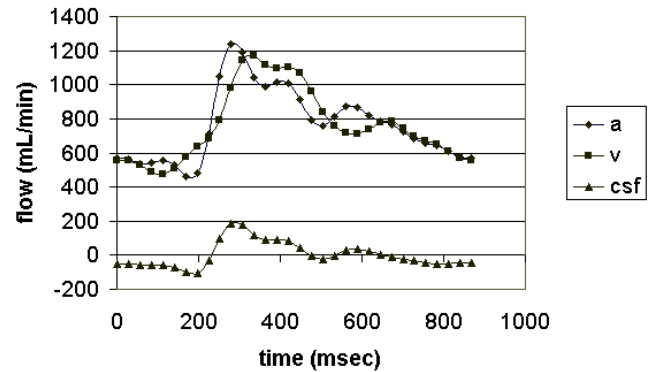
Fig. 3a: Arterial, venous, and CSF flow (healthy)

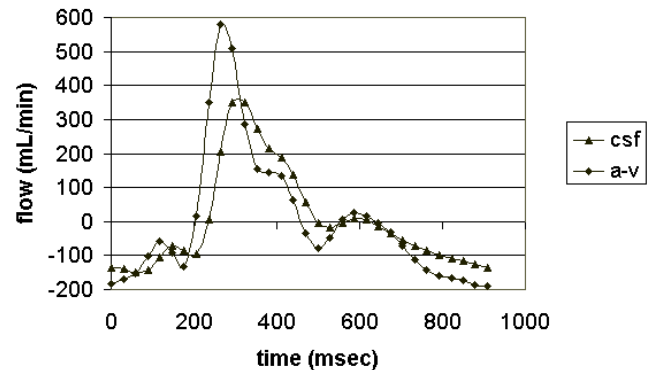Fig. 3b: Arterial, venous, and CSF flow (trauma)
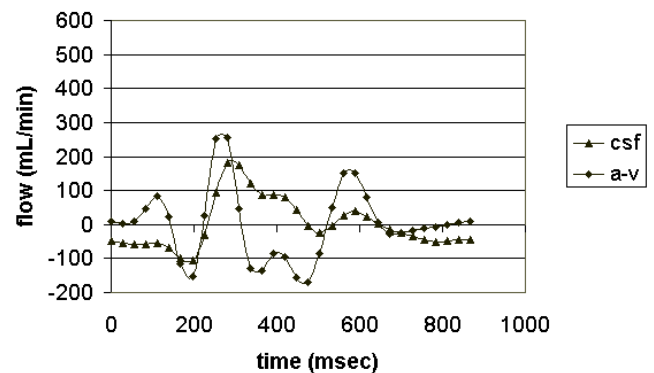

Fig. 4a: A-V, CSF (healthy)

Fig. 4b: A-V, CSF (trauma)

As a result, when the CSF waveform is subtracted from A-V for a healthy subject in order to calculate ICVC, the peak to peak of the volume waveform (0.49 mL) becomes smaller than the peak to peak of the volume of A-V waveform (1.08 mL) as shown in Fig. 5a. In the case of the trauma subject, the reverse is true. Because the A-V and the CSF waveforms are 'out of phase,' their subtraction becomes larger. Therefore, the peak to peak of the volume of A-V-CSF (0.61 mL) is actually larger than the peak to peak of the volume of A-V (0.36 mL), as shown in Fig. 5b.
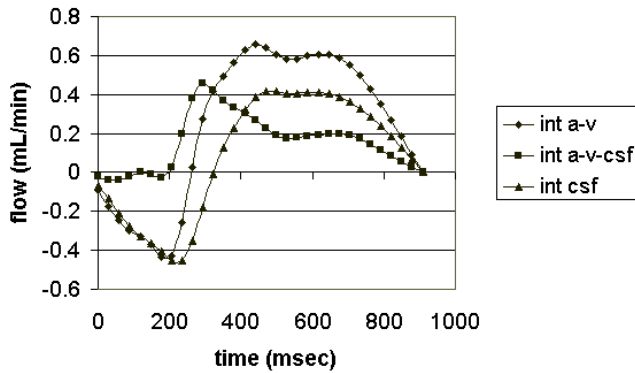
true as shown in Tables II and III where CSVC is always smaller than ICVC because of differing temporal information.

This anomaly appears to result from the increased pulsatility of the venous waveform. This may be due in part to decreased compliance in the venous channel or a direct link between arterial and venous flow channels, bypassing the capillaries and thus, any modulation that the brain tissue may offer (A-V shunt). The fact that there is net outflow of blood during the later part of systole is not explained by the possibilities mentioned.

Fig. 5a: Integrals of A-V, A-V-CSF, CSF (healthy)

Fig. 5b: Integrals of A-V, A-V-CSF, CSF (trauma)

Fig. 6: Volume change difference vs. pulsatility ratio

$$y = -1.7051x + 1.2205$$
$$R^2 = 0.7872$$
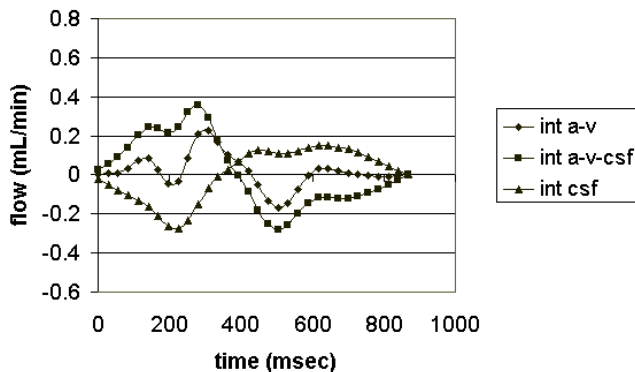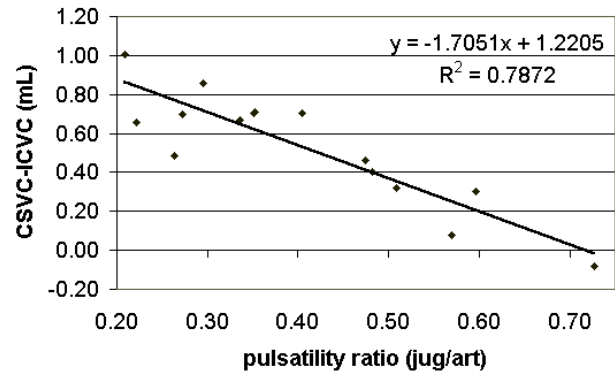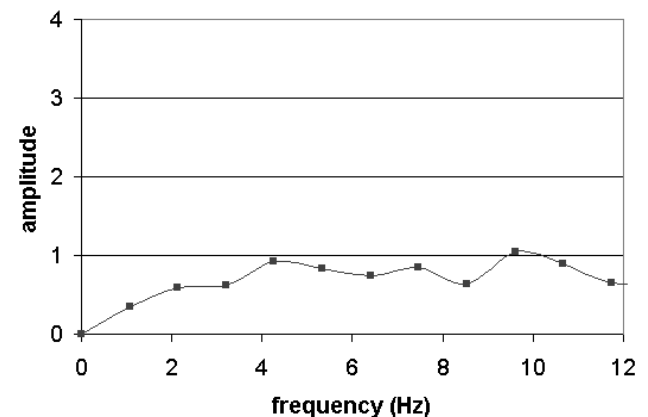

Fig. 7a: Modulation transfer function (healthy)
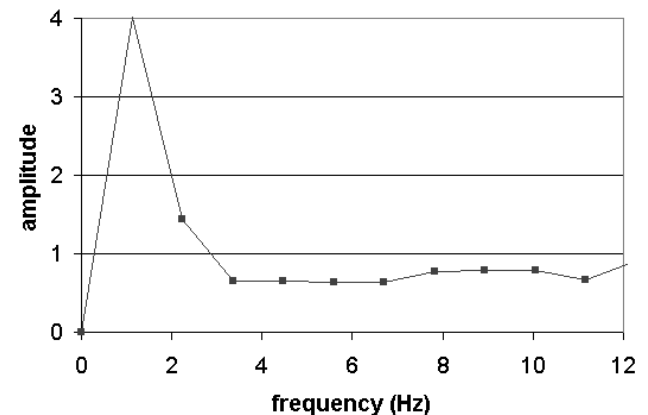
Fig. 7b: Modulation transfer function (trauma)

Venous pulsatility can be measured by the pulsatility ratio, which is defined as the ratio of the maximum amplitude of venous flow to the maximum amplitude of arterial flow. The difference between the CSVC and the ICVC is plotted against the pulsatility ratio for healthy normal subjects in Fig. 6. This graph demonstrates that as venous pulsatility increases relative to arterial pulsatility, the volume change difference goes down. The trauma cases that have been studied are cases in which the volume change difference has become less positive because of extremely high venous pulsatility.

It appears intuitive that the craniospinal volume change should always be larger than the intracranial volume change, which is the volume change in one of two components of the system. This is the case in healthy normal subjects as shown in Table I. In some subjects, including one that was originally studied as a normal reference, this consistently did not hold

A complementary way to characterize the craniospinal system dynamics is the application of modulation transfer function analysis using the A-V as the driving force (i.e. input) and the rate of intracranial volume change as the output. This approach has been previously described by Alperin, et al. [3]. An example of a modulation transfer function for a normal volunteer and for subject D who had experienced trauma are shown in Fig. 7a and 7b, respectively. The modulation transfer function of subject D shows resonance at the first harmonic (the heart rate in Hz). The "resonance" phenomenon is the result of the fact that venous blood outflow is larger than arterial inflow during systole and the fact that the CSF flow is out of phase with the A-V waveform in the same portion of the cardiac cycle. The authors are currently investigating possible mechanisms that may explain this behavior.

The authors speculate that checking for ICVC to be greater than CSVC could become an indicator of head trauma. Further studies are needed to evaluate the degree of difference between ICVC and CSVC and how it relates to the extent of the trauma.

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